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Epizootiology and Distribution of Transmissible Sarcoma in Maryland Softshell Clams, *Mya arenaria*, 1984–1988

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Seasonal and geographic studies of transmissible sarcoma in Maryland softshell clams, *Mya arenaria*, were carried out from 1984 to 1988. Three major epizootics occurred in our sampling location during this time, resulting in prevalences as high as 90%, with comparable mortalities in other high prevalence areas. The disease invaded populations of large adult clams first, later spreading to the small juvenile clam populations. An apparent 2-year cycle was noted with varying seasonal effects. Affected sites tended to be in the main stem of Chesapeake Bay north of Tangier Sound, primarily in the areas where the major harvesting occurs. Several sites, mostly in upstream locations, were consistently free of disease. The epizootiological study supports the interpretation that the disease is infectious exclusively to this species. Regression analysis between sarcoma prevalence and contaminant levels in clam tissues showed a significant correlation (p = 0.0001) between chlordane levels and this disease. No correlations were found with other contaminants that were analyzed.

Introduction

A neoplastic disease, called hematopoietic neoplasm (HN), in the softshell clam *Mya arenaria* was first reported from New England waters by several investigators (*1–4*), with several contaminants suggested as causative agents. Other studies demonstrated that the disease was transmissible (*5,6*). In 1984, an epizootic that was clearly new to this region (*7*) appeared in Chesapeake Bay softshell clams. In more recent studies the disease was designated as transmissible sarcoma because cell origin is still not definitively established, and additional work reconfirmed the transmissibility (*8*). Rare cases were diagnosed in 1979, 1981, and early 1983. In December of 1983 and early 1984, high prevalences were found in several Maryland soft clam populations which resulted in increased research effort. Previous studies have shown that the disease was originally confined to the Atlantic coastal area from the Hudson River drainage north. The disease was new to the Chesapeake Bay, and it probably was introduced from New England subsequent to the decimation of Chesapeake Bay stocks caused by Hurricane Agnes in 1972. The progressive malignant nature of the disease was demonstrated via laboratory studies using a new diagnostic method (histocytology) combined with a clinically significant staging system (*7*). The disease was transmissible from animal to animal by apparent transplantation of cells (*8*). A monoclonal antibody was developed against sarcoma cells from Massachusetts clams (*9*) that cross-reacted with Maryland clam sarcoma cells (*7*). A new monoclonal antibody was developed by R. Lundstrom, National Marine Fisheries Service Laboratory, Gloucester, Massachusetts (personal communication, 1988) against sarcoma cells from Maryland clams. Use of the antibody should result in increased diagnostic efficiency and could assist in determining the cell origin of the sarcomas.

While many studies of contaminants and contaminated locations have been reported [i.e., oil (*1,2,4*) and PCBs (*10*)], no clear correlations of pollution levels and occurrence of clam sarcomas have been demonstrated. For the present study, field and laboratory examinations of prevalences of soft clam sarcoma and clam mortality were initiated in 1984. Annual surveys of key geographic populations were conducted, as was monthly monitoring of adult and juvenile clams from Swan Point, Chesapeake Bay, Maryland, an area of continued high prevalence over the past 4 years (1984–1988). The Maryland Department of the Environment analyzed softshell clam tissues for contaminants taken during this time period from sites close to our histocytology survey locations (Tables 1–3). Seasonal and geographic sarcoma prevalences were compared with levels of contaminants for the presence or absence of correlations.

Materials and Methods

Sampling

Clams were collected using a commercial Hanks-type hydraulic conveyer belt clam dredge. Field estimations of mortality were done by counting the first 100 clams (live and dead)

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Table 1. Status of softshell clam sarcoma epizootic, 1984–1988.

Location	Code	Date	Mean length, mm	*n*	Sarcoma prevalence, %	Estimated mortality, %	Clams/ yd^2
Eastern Shore							
Tolchester		6/1/88	72.0	50	0	1	1.0
Piney Point	PP	9/17/85	58.5	50	0		
		3/10/86	60.1	42	0	0	1.5
		6/11/87	—	50	0	0	11.0
		10/8/87	—	50	0	6	3.1
		1/20/88	71.7	50	0	0	4.0
Ferry Point	FP	10/23/84	—	54	6	0	
		5/22/85	68.8	50	2	0	0.7
		9/17/85	49.2	30	10	2	
		3/13/86	57.6	40	5	4	1.1
		6/11/87	67.1	50	10	0	8.0
Parson Island	PR	10/23/84	—	50	14	0	1.9
		5/22/85	—	50	14	1	2.5
		9/9/85	—	34	50		
		3/6/86	59.6	40	10	8	1.3
		6/22/87	63.7	50	58	2	3.0
		5/5/88	72.0	50	60	15	0.7
Poplar Island	PI	5/4/84	—	50	42		
		9/9/85	57.2	50	67		
		3/1/86	55.5	41	2	0	1.0
		6/22/87	61.7	50	14	0	0.7
Kent Point	KP	9/7/85	54.0	50	33		
		3/1/86	59.8	41	2	0	1.2
		6/18/87	71.2	50	4	0	1.3
Wye River	WR	3/1/86	61.9	44	0	4	1.3
		6/22/87	66.1	50	10	0	5.0
Ranch House	RB	9/16/85	45.2	30	10		
		3/18/86	58.4	40	2	0	0.8
		6/18/87	74.0	50	6	0	4.0
Race Track	RT	3/14/85	65.8	54	0		
		9/30/85	50.3	30	0		
		5/9/86	68.9	44	0	0	1.0
		7/9/87	69.5	50	0	0	4.0
		5/25/88	77.0	50	0	2	0.6
Castle Haven	CH	9/30/85	41.5	30	10		
		5/9/86	64.7	44	0	0	0.3
		7/9/87	72.7	50	10	5	1.3
Bar Neck	BN	9/30/85	40.5	30	30		
		5/9/86	61.6	42	14	4	0.3
		7/9/87	70.2	50	0	3	2.0
Western Shore							
Bodkin Point	BO	5/23/88	75.0	50	0	3	1.3
Sandy Point	SA	9/23/85	49.2	30	10		
	SA	3/18/86	52.3	49	4	0	1.7
	SA	6/18/87	67.5	50	6	0	18.0
	SAJ	7/18/88	60.2	50	(2)	1	8.0
Three Sisters	TS	5/4/84	—	50	50		
	TS	9/2/85	41.5	30	3		
	TS	3/18/86	63.9	40	0	0	0.3
	TS	6/18/87	68.5	45	9	0	2.0
	TS	6/1/88	76.0	50	10	7	2.2
Brooms Island	BI	6/8/88	85.0	50	0	—	—

as they came up on the dredge belt. Numbers of clams/min were counted, and an estimate of density (clams/yd^2) was calculated using vessel speed and the width of the dredge. Salinity and temperature were determined at the site using a conductivity-type electronic salinometer.

Diagnosis

Samples of 30 to 50 live adult and juvenile clams were collected (Tables 1 and 2). Each clam was labeled with an indelible marker by sample code, date, and consecutive number. Clams were placed in flowing seawater until bleeding was accomplished. Methods described by Farley et al. (*7*) were used to produce fixed histocytological monolayer preparations from each clam. A preliminary live diagnosis was done at the time of bleeding. The preparations were then fixed in modified McDowell's fixative (1G4F) (*11*) and stained with Feulgen picromethyl blue stain (*12*) for a more accurate diagnosis. Histocytology was the standard method of diagnosis for all samples. Sarcoma stages were determined using a newly modified system on the basis of the ratio between normal hemocytes and sarcoma cells: stage 1 was 1 to 9 cells/100,000; stage 2 was 1 to 9 sarcoma cells/10,000 cells; stage 3 was 1 to 9

Table 2. Swan Point monthly data, 1986, 1988.

Date	Mean length, mm	n	Prevalence, %, Stage 1	2	3	4	5	6	7	Total, %	Mortality, %	Clams/yd^2	Salinity, ‰	Temperature, °C
Adult population (code SWA)														
3/10/86	57.9	50								0	0	0.3	—	—
4/18/86	62.9	76			1	1				2	1	0.4	—	—
5/19/86	63.7	52		2		11				13	4	0.3	—	—
6/14/86	65.6	50		4	4	6				14	3	0.5	—	—
7/10/86	67.2	48	3	6	13	6				29	5	0.3	—	—
8/8/86	69.3	50	4	4	8					16	12	0.3	—	—
9/25/86	70.3	50	8	8	10	10		4		40	6	0.1	—	—
10/27/86	71.9	52	6	6	2					22	14	0.1	—	—
11/24/86	62.3	50	2							2	1	—	—	—
12/19/86	63.9	60	2	2	2		2			8	0	8.0	10.5	10.0
1/14/87	60.4	50	6	2						8	0	8.0	10.5	9.5
2/23/87	60.1	50	8							8	0	12.0	10.5	9.5
3/23/87	61.9	60			2					2	0	11.0	13.5	9.0
4/20/87	64.9	50	2		2					4	0	14.0	11.8	20.0
5/27/87	65.6	50	6	2	10					18	0	14.0	—	—
6/11/87	67.8	50	4	4	20	2				30	0	13.0	—	—
7/16/87	62.4	60	2	14	12	2	16	8		54	3	13.0	—	—
8/11/87	66.9	50	10	2	4		2			18	2	10.0	—	—
9/8/87	66.9	50	6	2						8	1	9.0	—	—
10/13/87	66.4	50	20	16	4		8	6	2	56	7	2.8	—	—
11/18/87	65.7	50	8		2	14	10	22	29	58	9	3.6	14.0	13.0
12/14/87	65.7	50	20	8		2	6	6	22	66	4	4.6	11.0	—
1/25/88	67.0	50	12	6		14	6	16	6	60	7	2.4	9.5	—
2/17/88	69.5	50	8	4	2	12	8	16	14	66	2	1.5	—	—
3/21/88	70.9	50	6	4	4	4	10	24	8	60	1	3.0	7.5	—
4/22/88	68.0	50		4	4	4	16	8	8	44	2	1.7	—	—
5/20/88	71.9	50	2		2	2	8	6	22	42	10	2.2	8.0	19.0
6/13/88	70.7	50	8		2	8	4	34	16	72	40	0.5	8.0	20.0
7/12/88	67.9	50	2	4	4	10	8	6	4	38	35	0.6	9.8	26.0
8/3/88	72.4	50								0	20	0.2	9.2	28.0
9/13/88	70.5	50								0	36	0.1	10.0	24.0
Juvenile population (code SWJ)														
5/19/86	36.7	43								0	0		—	—
6/14/86	41.9	50								0	0		—	—
7/10/86	42.5	40		4	3	3				10	0		—	—
8/8/86	42.9	50				2				2	0		—	—
9/25/86	51.1	50	8	2		4				14	0	10.0	—	—
10/27/86	54.0	60	2	6						8	0	12.0	—	—
11/24/86	51.3	50		2						2	0	4.0	—	—
8/11/87	38.5	50								0	0	—	—	—
9/8/87	38.9	50	4							4	0	—	—	—
10/13/87	43.2	50	8							8	1	8.5	—	—
11/18/87	45.9	50	2					2		4	0	8.0	14.0	13.0
12/14/87	43.0	50	2							2	0	12.0	11.0	—
1/27/88	48.7	50	4							4	0	12.0	9.5	—
2/18/88	48.2	50	8		2					10	0	14.0	—	—
3/23/88	51.8	50				2				2	0	18.0	7.5	—
4/22/88	53.6	50	2					2		4	0	11.4	—	—
5/20/88	54.8	50								0	1	12.0	8.0	19.0
6/14/88	54.3	50	10	2				2	2	16	2	7.8	8.0	20.0
7/13/88	56.2	50	12	8	14	11	12			76	0	14.5	9.8	26.0
8/17/88	55.2	50								0	4	12.0	9.2	28.0
9/13/88	56.6	50								0	13	15.0	10.0	24.0

sarcoma cells/1000; stage 4 was 1 to 9% sarcoma cells; stage 5 was 10 to 49% sarcoma cells; stage 6 was 50 to 89% sarcoma cells; and stage 7 was 90 to 100% sarcoma cells.

Contaminant analyses for an array of inorganic and organic contaminants were conducted by the Maryland Department of the Environment from clam tissue samples collected at various times during the period of study (Table 3) (M. J. Garreis, personal communication, 1988).

Results

A map of the Maryland portion of Chesapeake Bay shows the location of sites positive for sarcoma and negative sites (Fig. 1). Table 1 shows the disease prevalence, mortality, clam population density, and locations (Fig. 1) surveyed from 1984 to 1988. Swan Point data (*10*) are presented separately (Table 2). In these surveys, Parson Island, Poplar Island, Kent Point, Bar Neck, and Three Sisters all had periods of very high (> 40%) prevalences.

Table 3. Analysis of Chesapeake Bay softshell clam tissues for sarcoma prevalence and contaminant levels.

Location	Peak sarcoma prevalence, %	Contaminant levels, ppm[a]												Contaminant sample date
		Cu	Zn	Pb	Hg	Cd	Cr	As	PHC alpha	DDT	Dieldrin	Heptachlor epoxide	Chlordane	
Race Track	0	3.87	16.90	0.70	0.004	0.30	0.5	0.04	0.001	0.02	0.006	0.003	0.02	10/5/83
Brooms Island	0	4.76	20.80	0.90	0.001	0.50	0.6	0.85	0.001	0.02	0.006	0.003	0.01	4/30/85
Bachelors Point	0	8.10	19.70	0.40	0.006	0.12	0.2	0.04	0.001	0.02	0.020	0.003	0.03	5/17/78
Piney Point	0	6.73	15.40	0.70	0.007	0.71	0.7	1.81	0.002	0.01	0.006	0.003	0.02	10/7/85
Wicks Beach	0	4.81	15.20	0.40	0.006	0.14	0.1	1.90	0.002	0.01	0.006	0.003	0.03	5/8/85
Sandy Point	10	11.50	39.20	0.40	0.006	0.30	0.1	0.96	0.005	0.02	0.006	0.003	0.03	5/17/82
Ferry Bar	10	9.59	38.80	2.30	0.003	0.96	1.1	0.48	0.001	0.01	0.006	0.003	0.02	10/7/85
Thomas Point	23	11.70	21.70	0.40	0.004	0.10	0.4	0.94	0.003	0.01	0.006	0.003	0.03	9/28/83
Three Sisters	40	10.20	51.30	1.10	0.005	0.36	0.9	0.64	0.011	0.01	0.006	0.011	0.06	8/19/80
Parson Island	60	8.44	19.70	0.80	0.011	0.39	0.8	0.47	0.002	0.01	0.006	0.003	0.08	9/28/83
Swan Point	76	8.22	21.90	1.70	0.007	0.52	0.4	1.3	0.009	0.01	0.015	0.006	0.07	9/29/83
	X	Y_1	Y_2	Y_3	Y_4	Y_5	Y_6	Y_7	Y_8	Y_9	Y_{10}	Y_{11}	Y_{12}	
R[b]		0.37	0.18	0.38	0.52	0.07	0.23	0.03	0.66	0.51	0.17	0.49	0.96	
p		0.27	0.59	0.25	0.085	0.85	0.50	0.93	0.029	0.11	0.6	0.13	0.0001[c]	

[a]Contaminants that occurred at levels below 0.01 ppm were not included in the table.
[b]Regression analysis was performed using sarcoma prevalence as X and contaminant concentrations as Y_n.
[c]Only chlordane exhibited a highly significant correlation.

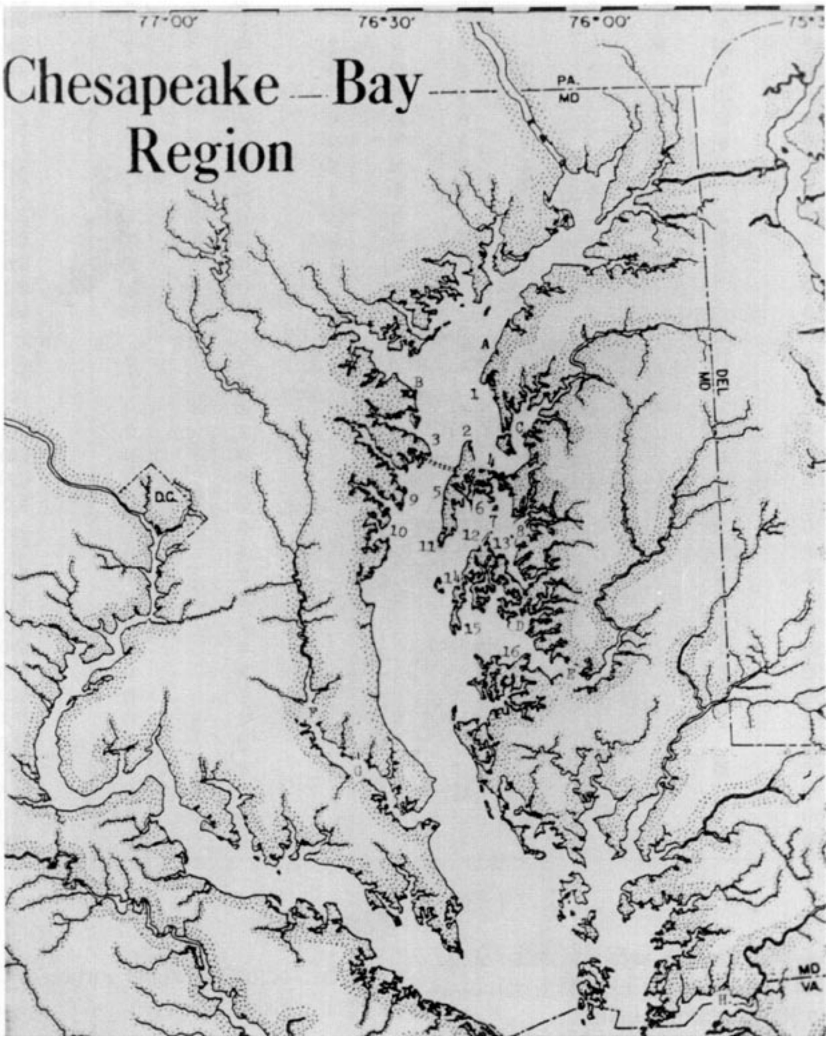

FIGURE 1. Chart of the Maryland portion of Chesapeake Bay. Numbered sites are locations where sarcomas have been diagnosed in clam populations. Lettered sites are locations where clam populations have heen consistently negative for the disease. Sites are as follows: (1) Swan Point, (2) Ranch House, (3) Sandy Point, (4) Ferry Bar, (5) Brick House, (6) Eastern Bay, (7) Parson Island, (8) Wye River, (9) Thomas Point, (10) Three Sisters, (11) Kent Point, (12) Claiborne, (13) Miles River, (14) Poplar Island, (15) Bar Neck, (16) Castle Haven, (A) Tolchester, (B) Bodkin Point, (C) Piney Point, (D) Oxford Lab, Tred Avon River, (E) Race Track, (F) Buzzards Island, (G) Brooms Island, (H) Marumsco Bar.

Table 4. Epizootic neoplasia in marine bivalve mollusks.

Area	Date	Prevalence	Percent	Type	Condition	Reference
Macoma balthica						
Chesapeake Bay sites						
Wells Point	3/70	5/50	10	Gill carcinoma	Trace of mirex	(*13*, unpublished)[a]
Fox Hole	5/71	8/100	8	Gill carcinoma		(*13*,unpublished)
Double Mills	5/71	0/100	0	Gill carcinoma	Light domestic sewage	(*13*, unpublished)
Quonsett Point, RI	3/71	0/100	0	Gill carcinoma	Navy dump (hydrocarbons)	Unpublished
Mytilus edulis						
Yaquina Bay, OR	2/69	3/73	10	Sarcoma	Unknown	(*14*)
Yaquina Bay, OR	1/81	40/200	20	Sarcoma	PAH	(*15*)
Coos Bay, OR	Winter/72	1/50	2	Sarcoma	Unknown	Unpublished
Tillamook Bay, OR	Winter/72	1/50	2	Sarcoma	Unknown	Unpublished
British Columbia, Canada sites						
Departure Bay	1/78	1/50	2	Sarcoma	Unknown	Unpublished
Cowichan Bay	12/81	7/24	29	Sarcoma	Marina, chemical wastes	(*16*)
Hatch Point	1/81	4/24	16	Sarcoma	Clean	(*16*)
Puget Sound, WA	11/86	8/50?	40	Sarcoma	Unknown	(*17*)
United Kingdom (7 in 21 sites positive)						(*18*)
Heysham	4/78	6/200	3	Sarcoma	Clean	(*18*)
Denmark (3 sites, all positive)						(*19*)
Hindsgavl	12/83	2/300	2.3	Sarcoma	Contaminants not noted	(*19*)
East Coast of the United States, *Virginia to Canadian border*						(*20*)
	Multiyear study	0/700			All gradations of contamination	(*20*)
Booth Bay Harbor, ME	1985?	1/?			?	(*21*)
Boston Harbor, MA	1986?	1/30			Heavily contaminated	(22)
Long Island Sound, NY	1986?	1/30?			Moderately contaminated	(22)
Ostrea lurida						
Yaquina Bay, OR	Winter/70	20/50	40	Sarcoma	PAH?	(*23*)
Macoma (3 species)						
Yaquina Bay, OR	Spring/70	3/150	5	Sarcoma	PAH?	(*24*)
Cerastoderma edule						(25)
Cuskinny, Ireland	6/84	15/28	53	Sarcoma	Sewage?	(25)
Mya arenaria						
Annisquam River, MA	9/72	10/50	20	Sarcoma	PSP (red tide)	(*24*, unpublished)
Warpswell Neck, ME	9/72	7/31	22	Sarcoma?	JP4	(*1*)
Searsport, ME	10/75	13/100	13	Gonadal neoplasm	#2 Fuel oil, JP5	(*1*)
Searsport, ME	10/75	2/100	2	Sarcoma	#2 Fuel oil, JP5	(*1*)
Searsport, ME	?	?	?	Gonadal	2,4-D; 2,4,5-T	(*26*)
Sandy Point, RI	1/76	2/10	20	Sarcoma	Nominally polluted	(*3*)
Quonsett, RI	9/76	18/45	40	Sarcoma	Hydrocarbons (Navy Dump)	(*3*)
Bourne, MA	9/76	27/60	45	Sarcoma	#2 fuel oil	(*3*)
Greenpond, MA	2/78	14/51	28	Sarcoma	No known contaminant	(*3*)
New Bedford, MA	3/83	4/30	13	Sarcoma	PCB, heavy metals	(*10*)
Stonington, CT	1/84	13/29	45	Sarcoma	Not presented	(*27*)
Westport, CT	1/85	18/30	60	Sarcoma	Not presented	(*24*)
Swan Point, MD	8/85	45/50	90	Sarcoma	Nominal pollution	(*28*)

[a]"Unpublished" denotes unpublished data from previous National Marine Fisheries Services Oxford Laboratory notes.

Ferry Bar, Wye River, Ranch House, Sandy Point, Thomas Point, and Castle Haven had low prevalences. Samples from Tolchester, Bodkin Point, Piney Point, Race Track, and Brooms Island were consistently negative.

Table 2 shows the average clam size, sarcoma prevalence by stage, mortality, and population density in adult and juvenile clams collected monthly from Swan Point, upper Chesapeake Bay, from March 1986 through August 1988. Salinity and temperature at time of collection are also listed for certain of the samples. Three epizootics have occurred at Swan Point during this period; the prevalence went from 0 to 40% from March to September 1986 in the adult population sampled (> 56 mm). Prevalence dropped after that, presumably due to mortality, and then increased again in surviving younger clams (62.3 mm) (which then became the adult population beginning in November 1986) beginning in April 1987 and peaking at 54% in June. Prevalence dropped during the summer but increased again in the fall, finally peaking at 72% in June of 1988, with field evidence of mortality during this time. Prevalence dropped to 36% in July and to 0% in August and September. Prevalence in juvenile clams showed seasonally similar but much lower activity, peaking at only 14% in September of 1986 and 10% in February of 1988. Prevalence increased dramatically from 16% to 76% in July 1988 (1 month after the mass mortality and high prevalence seen in the adult population), but dropped to 0% in August and September of 1988.

Table 3 shows contaminant levels in clam tissues taken from the study sites as well as regression analysis data between sarcoma prevalence and tissue levels. Copper and zinc were highest in clams from western shore sites (Sandy Point and Three Sisters)

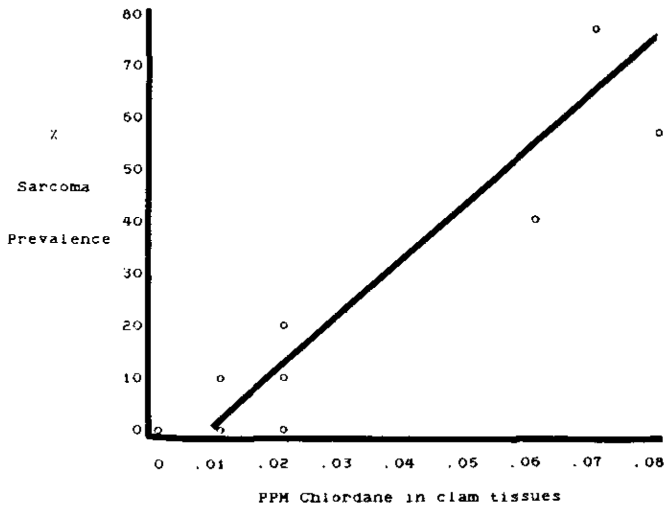

FIGURE 2. Scattergram of points by sample location comparing softshell clam sarcoma prevalence and chlordane burden in clam tissues in ppm. $R = 0.93$, $p = 0.0001$, intercept = -0.199; slope = 10.936; lower 95% confidence interval (mean) = 0.125; upper 95% confidence interval (mean) = 0.273.

and also at Kent Narrows (Ferry Bar). Swan Point and Brooms Island clams had high cadmium levels. Higher levels of dieldrin were seen in the Swan Point (epizootic) and Bachelors Point (sarcoma free) samples. Regression analysis (Fig. 2) showed a highly significant ($p = 0.0001$) correlation between sarcoma prevalence and chlordane tissue burdens, but correlations were not evident with other contaminants.

Discussion

An impressive literature has accumulated on epizootic neoplasia of bivalve mollusks in the past 20 years. Table 4 (*13–28*) is an attempt to summarize some of the data that are pertinent to the question, Is there a relationship between epizootic neoplasia and carcinogenic environmental contaminants in bivalve mollusks? The answer is still elusive. There is little evidence that any of the bivalve epizootics are associated with obvious environmental carcinogens, other than the correlations found in Yaquina Bay mussel sarcomas with PAH (*15*) and the chlordane correlation with Mya sarcomas that we are reporting here.

The course of the epizootic clam disease in Chesapeake Bay has been monitored since 1984. Epizootic disease has consistently remained in the main stem portion of the Bay while sites with lower prevalences were more common in mid-regions of the estuaries. Sites that have remained negative for sarcomas all occur in upstream locations. These are regions where freshwater influence is greatest; they are characterized by lower salinities, lower pH, and often higher pollutant levels (*29*). Changes in sarcoma prevalences have occurred from year to year that make interpretations very difficult. When the disease first struck the population in late 1983, high prevalences were confined to clams greater than 65 mm, with most cases found in animals larger than 70 mm. By late spring 1984, prevalences dropped to 0, concurrent with mortality. The disease was observed in the fall of 1984 in slightly smaller clams. Mortality followed the high 60% prevalence seen in the winter of 1984–1985, but new cases continued to occur throughout the spring and summer of 1985. The disease invaded juvenile clams in June 1985 (37% prevalence, which intensified to 70% by late August). Prevalences decreased to 0 with mortality in September 1985. The epizootic then subsided until 1986 when the cycle of disease and mortality seemed to repeat the 1983–1984 situation, with larger clams being affected. There has been some slight indication from recent observations that remissions may occur. This indication should be examined carefully because it suggests the development of resistance in challenged populations or environmental changes that may affect survival of neoplastic cells.

This epizootic has occurred in Maryland waters considered to be clean and safe enough for commercial harvest of shellfish. While there is evidence that significant levels of some heavy metals do exist in some areas sampled, no correlations are evident that link the clam sarcoma to contaminant levels, except in the case of chlordane. Based on mammalian experiments, this pesticide is considered to be carcinogenic; it is very persistent, and it is used widely for termite control. There is a clear straight-line relationship between chlordane tissue concentrations and prevalence levels of clam sarcomas in Chesapeake Bay populations. This new information suggests a possible cause-and-effect relationship between this disease and the pesticide and exacerbates concerns regarding the presence of this known carcinogen in tissues of clams and its effects. Further experimental studies are warranted. However, previous studies of this disease in Mya have experimentally demonstrated transmissibility in the absence of contaminants (*5,8*), and field studies discussed in this paper and previously (*7*) tend to reduce the likelihood of contaminant involvement in the etiology of this disease.

The possibility that other molluscan diseases are infectious has been demonstrated recently (*17,25*). Since other bivalve mollusks living in the same waters are not experiencing epizootic neoplastic disease [i.e., oysters, hard clams, mussels of several species, duck clams (Macoma)], this and other molluscan neoplastic diseases seem to be exclusively species specific, and all of them may prove to be transmissible diseases. Softshell clam sarcoma may be transmitted by transplantation of cells from animal to animal and may not require an infectious organism such as a virus. Some evidence already exists suggesting this possibility. The shift of epizootic prevalences from large clam populations to small clam populations, the experimental evidence for transplantation (*8*), and the lack of obvious viral infections in ultrastructural studies (*7*) all support this concept.

Conclusions

Epizootic manifestations of clam sarcoma (geographic spread, size versus prevalences) suggest that this is a transmissible disease. It can be postulated that when infective particles are scarce, the large clams become infected, presumably because the larger animals filter more water and have a greater likelihood of becoming infected. When severe outbreaks of disease occur in large clams, infective particles may be released in large numbers, thereby transferring the disease to the juvenile population, which then shows a similar epizootic pattern.

The first sarcoma outbreak in 1984 and 1985 resulted in economically significant mortalities that resulted in a scarcity of clams and higher prices. Since then, populations have returned to high levels, and the impact of the disease has lessened. The prevalences in the populations now (1988) have the potential of causing a recurrence of the 1984–85 devastation, possibly as early as 1989.

Chemical analyses by the Maryland Department of the Environment of clam tissues from sample sites in question do not indicate that levels of contaminants (with the exception of chlordane) are present that would play a role in the etiology of this disease. Chlordane might act directly or synergistically to enhance the development of this disease by either inducing or affecting the defense mechanisms that may protect clams from such diseases. Studies of epizootic neoplasia in other species of bivalve mollusks (oysters, clams, mussels) and in other areas do not show clear relationships between contaminants and neoplasia; in fact, infectious or transmissible sarcomatous diseases have been demonstrated in mussels (*17*) and cockles (*25*).

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